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Opinion Article

Role of Inflammatory and Pain Modulators in Treatment of Various Diseases

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DESCRIPTION

Most pain information originates from simple, bare nerve endings called nociceptors. Nociceptors form functional pain units with capillaries and mast cells in nearby tissues. Tissue injury causes depolarization of these nerve endings. This results in sensory impulses that propagate along the entire afferent fiber and reach the spinal cord. This stimulation of primary afferent fibres at the site of tissue injury triggers the axonal release of vesicles containing neuropeptides. These neuropeptides acts on autocrine and paracrine ways to sensitize nociceptors and increase their rate of stimulation. Cell damage and inflammation increase the levels of other chemical messengers such as histamine, bradykinin and prostaglandins around functional pain units. These additional mediators act synergistically to enhance the transmission of nociceptive impulses along sensory afferent fibres. Primary fibres migrate from the periphery to the dorsal horn, where they form synapses with secondary neurons and interneurons. Activation of interneurons has an inhibitory effect on further pain signaling. Efferent supraspinal impacts, in turn, determine interneuron activity by releasing various neurotransmitters, resulting in potent modulation of nociception. These phenomena involve many chemical mediators and receptor systems and may enhance pain perception qualitatively, quantitatively, temporally and spatially. The complexity and plasticity of the nociceptive system can complicate clinical pain management. Understanding the structural and chemical signals associated with this system can improve the use of existing analgesics and provide targets for the development of new, more specific analgesics.

Inflammation is a homeostatic response by angiogenic tissue. Nervous system either central or peripheral disease or injury can cause Neuropathic Pain (NP) and also alter the mediation of neuroinflammatory processes. Such conditions can cause pain unrelated to the toxic substance or painful stimulus, and inflammation unrelated to the presence of the toxic substance or the need to remove it. NP is the result of incomplete regeneration of the peripheral nervous system or Central nervous system. Inflammation is an important part of attempts to repair tissues/cells and may be regulated by an intact or defective nervous system. The initial cause of inflammation itself may or may not be the same as that of NPs and interplay of inflammatory and neuropathic processes inevitably occurs. Depending up on the resolution time, inflammation can be acute or chronic. In acute inflammation, vascular changes result from the accumulation of fluid and polymorphonuclear cells at the site of injury. Migration of hematopoietic elements appears to be directed by concentrations of prostaglandins that alter the outer chain affinity of adhesion molecule receptors. Acute inflammatory responses are systemic and include hyperthermia, leukocytosis, protein catabolism, and hepatic plasma protein synthesis such as C-reactive protein. Chronic inflammation is characterized by the simultaneous presence of cellular infiltration (lymphocytes and macrophages) and injury and repair. Pain is one of the first four classic signs of inflammation, along with redness, swelling, and heat. They are now recognized to be triggered by a series of chemically mediated events such as Changes in local vascular flow and patency, leukocyte infiltration. However, when inflammation becomes chronic, tissue is destroyed and the fifth most detrimental inflammatory signal, loss of function can emerge. The inflammatory process is multi-media which shows different responses depending on the nature of the noxious agent, the predominant cell type of some infiltrates and the involvement of various chemical mediators. In general, pain is an unpleasant sensation and a nociceptive sensation. Pain is not an isolated event, but a series of linear and nonlinear events, leading to a state of defensive hyperactivity, increasing the likelihood of recognizing new styles and optimizing the defense system. A situation of neuronal hyperactivity due to dysfunction of the somatosensory system can lead to neuropathic pain, sometimes co-occurring with inflammation. The resulting NPs are generated and maintained through a cascade of inflammatory processes.

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